are subpopulations of lymphocytes with different functions, antigen specificities and surface characteristics. The current thrust of much investigation is to identify and characterize these subpopulations.

JOHN W. PARKER, MD

REFERENCES

Rowlands D, Daniell R: Surface receptors in the immune response. N Engl J Med 293:26-32, Jul 3, 1975

Greaves M, Owen J, Raff M: T & B lymphocytes. New York, American Elsevier Publ Co, 1974

Hansen JA, Good RA: Malignant disease of the lymphoid system in immunological perspective. Hum Pathol 5:567-599, Sep 1974

Immune Deficiency in Adults

INITIALLY, immune deficiency was classified as either primary (congenital) or secondary. The primary form was generally considered to occur in infants and children. The secondary form, seen in adults, was due to another disease process, such as protein loss in renal or gastrointestinal disease producing hypogammaglobulinemia, or chronic lymphocytic leukemia (CLL). However, as awareness of the various deficiency states increased, some adults were found to have hypogammaglobulinemia or defects in their cellular immunity without evidence of the classically associated diseases such as CLL or Hodgkin's disease. These patients were reported to have late onset or primary acquired immunodeficiency as adults —recent literature refers to this as common variable immunodeficiency. Either arm (humoral or cellular) of the immune system may be involved, although most cases reported involve the humoral aspect. Common variable immunodeficiency is relatively common, and both men and women may be affected. A familiar pattern is not usually found. One common manifestation is recurrent infection of the sinopulmonary tract, and frequent episodes of pneumonia may lead to chronic bronchiectasis and severe respiratory insufficiency. Sinus and ear infections may lead to diminished hearing. Another manifestation is a sprue-like syndrome with a wide range of malabsorption difficulties, and gastrointestinal tissues in some cases show nodular lymphoid hyperplasia. Tissues of various organs may show noncaseating granulomas, and there may be generalized hyperplasia of the reticuloendothelial system. A patient's serum may contain a wide variety of autoantibodies.

Diagnosis is usually made by quantitation of serum immunoglobulins and evaluation of cellular immunity—that is lymphocyte count and skin tests. Determination of the number of B-cells in peripheral blood—a more sophisticated test—was not always helped in the evaluation of hypogammaglobulinemia, as the number of cells varies from 0 to "normal." In some cases where the B-cell number has been "normal," there is evidence of a suppressor effect by T-cells. Therefore it appears that all variations of production and secretion may be found, so care should be taken in making the diagnosis and assessing individual cases.

STEBBINS B. CHANDOR, MD

REFERENCES

Rosen F: Primary immunodeficiencies. Pediatr Clin North Am 21:533, Aug 1974

Waldman TA, Durm M, Broder S, et al: Role of suppressor T cells in pathogenesis of common variable hypogammaglobulinemia. Lancet 11:609, Sep 14, 1974

Amniotic Fluid Protein of the Second Trimester

SECOND TRIMESTER transabdominal amniocentesis and subsequent laboratory examination of the constituents of the amniotic fluid is of established value in the identification of fetal morbidity.

Amniotic cell cultures with specific enzyme assays is of diagnostic value in over 20 inborn defects. High levels of amniotic fluid alpha-fetoprotein (AFP) are now considered a good marker of neural tube defects such as anencephaly and spinal bifida. This observation of elevated AFP is most valid and of pragmatic value between 14 and 18 weeks gestation when diagnosis of a major developmental neural abnormality is to be followed by abortion. Additional amniotic proteins, especially the immunoglobulins, have not yet been completely evaluated. However, some measurements are known. It is possible to identify and quantitate albumin, immunoglobulin G (IgG), transferrin and alpha-1 antitrypsin in "normal" midtrimester amniotic fluid specimens with the use of commercially available single radial immunodiffusion plates and monospecific antisera.

Second trimester amniotic fluid also contains immunoglobulin A (IgA), Gc globulin and alpha fetoglobulin. Immunoglobulin M (IgM) and alpha-2-macroglobulins are not detected in normal fluids with commercially available reagents. IgG does cross the placenta but the other immunoglobulins apparently do not. Therefore, the